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Health and Environmental Effects
Profile for Dichloroprop

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FOR DICHLOROPROP

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16. ABSTRACT The Health and Environmental Effects Profile for <u>dichloroprop</u> was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents. Quantitative estimates have been presented provided sufficient data are available. <u>Dichloroprop</u> has been determined to be a systemic toxicant. An Acceptable Daily Intake (ADI), defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect, for <u>dichloroprop</u> is 2.5×10^{-2} (mg/kg bw/day) for oral exposure.		
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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEP's are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEP's will become part of the EPA RCRA docket.

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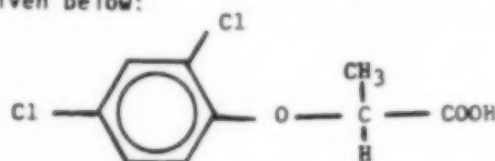
LIST OF ABBREVIATIONS

BCF	Bioconcentration factor
bw	Body weight
DNA	Deoxyribonucleic acid
GI	Gastrointestinal
i.p.	Intraperitoneal
LC ₅₀	Concentration lethal to 50% of recipients
LD ₅₀	Dose lethal to 50% of recipients

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Dichloroprop is the common name for 2-(2,4-dichlorophenoxy) propanoic acid ($C_9H_8Cl_2O$, m.w. 235.1). It is also known as 2,4-DP, 2-(2,4-DP) and α -(2,4-dichlorophenoxy) propionic acid. Some of the trade names of this compound include Celatox-DP, Cornox RK, Desormone, Hedonal DP, Herbizid DP, Hormatox, Kildip, Polymone, Polytox and Weedone DP (CIS; SANSS Data Base, 1983). Dichloroprop exists in two optically active forms. Only the (+)-isomer shows biological activity. Commercial products contain approximately equal amounts of the two isomers (BCPC, 1977). The structure of dichloroprop is given below:



The Chemical Abstract Services (CAS) Registry number for this compound is 120-36-5.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Pure dichloroprop is a colorless, odorless, crystalline solid (BCPC, 1977). The commercial grades may be white to tan in color with a faint phenolic odor (BCPC, 1977; WSSA, 1979). Some of the physical properties of dichloroprop (free acid) are given below (BCPC, 1977; WSSA, 1979 unless otherwise stated).

Melting point:	117.5-118.1°C (pure product) 114°C (technical product)
Density (temperature unspecified):	-1.42
Solubility:	350 mg/l in water at 20°C 595,000 mg/l in acetone at 28°C 85,000 mg/l in benzene at 25°C

pK_a ($-\log_{10}$ of
dissociation constant):

2.86 at 22-26°C (Cessna and
Grovers, 1978)

K_{oc} (soil sorption coefficient
standardized with respect to
organic carbon content) (calcu-
lated by regression equation
from water solubility data):

170 (Kenaga, 1980)

BCF (calculated by regression
equation from water solubility
data):

23 (Kenaga, 1980)

The vapor pressure data for the free acid form of dichloroprop are not available, but the pressure is expected to be very low (BCPC, 1977; Bovey, 1980). The free acid is stable toward heat and resistant toward hydrolysis and atmospheric oxidation and reduction reactions under normal conditions (BCPC, 1977).

1.3. PRODUCTION DATA

Dichloroprop is commercially produced by the reaction of 2-chloropropionic acid with 2,4-dichlorophenol in alkaline solution of $>pH$ 9 (WSSA, 1979). The three domestic producers of dichloroprop listed by SRI (1983) are Rhone-Poulenc Inc., Portland, OR; Union Carbide Corp., St. Joseph, MO; and Vertac Chem. Corp., Jacksonville, AR. However, according to USITC (1983), the two U.S. manufacturers of this chemical are Diamond Shamrock Corp., Tuscaloosa, AL, and Fike Chemicals Inc., Nitro, WV. Specific production data for this compound could not be obtained from the available literature. In 1982, the total U.S. production of dimethylamine salt and isooctyl ester of 2,4-D, the 2,4-D compounds of largest usage, was <20 million pounds (USITC, 1983). Therefore, the total annual U.S. production of dichloroprop may be <20 million pounds.

1.4. USE DATA

Dichloroprop is a translocated postemergence herbicide used in the control of weeds in small grain cereals. In combination with other herbicides such as MCPA, benazolin and dicamba, it is an effective broad spectrum herbicide. Dichloroprop is also used for brush control in nonagricultural land (WSSA, 1979).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

Limited data pertaining to the fate and transport of dichloroprop in aquatic media are available in the literature. In subject areas where information gaps exist, the fate and transport of dichloroprop have been speculated in the following discussion to resemble the aquatic behavior of a very similar compound, 2,4-D.

The chemical process most likely to significantly affect the aquatic fate of dichloroprop esters is hydrolysis; however, no comprehensive study on the rate of hydrolysis of dichloroprop esters in water has been made. The hydrolysis of 2,4-D esters, on the other hand, has been studied by several authors. Since the hydrolysis constants are inversely proportional to the dissociation constants, the rates of hydrolysis for 2,4-D and dichloroprop esters are expected to be quite similar because the dissociation constants of the two free acids are similar (pK_a of 2.87 vs. 2.86) (Cessna and Grover, 1978). If the hydrolysis rates for dichloroprop and 2,4-D esters are assumed to be similar, chemical hydrolysis may play an important role in determining the fate of dichloroprop esters in basic natural waters; however, in neutral or acidic water, its contribution will be minimal (Zepp et al., 1975; Mill, 1980). For example, the estimated half-lives for methyl, isopropyl and n-butyl esters of 2,4-D of 44, 710 and 220 days, respectively, at pH 6 (Zepp, et al., 1975) may be close to the half-lives of the dichloroprop esters. The corresponding half-lives at pH 9 were estimated to be 1.1, 17 and 5.2 hours (Zepp et al., 1975). Although the hydrolysis of dichloroprop esters will produce the free acid and not any decomposition products, the hydrolytic process may be important in determining the fate of dichloroprop esters in aquatic media. For example, the rate

of volatilization and microbial degradation in aquatic media may be strongly dependent on the nature of dichloroprop species (acid, salts or esters) present in water. If the hydrolytic process is substantially faster compared with other fate determining processes, it is the free acid and not the esters that may determine the fate of dichloroprop in aquatic media.

The second process that may contribute significantly to the disappearance of dichloroprop from aquatic media is photolysis. Although the photolytic behavior of dichloroprop under natural sunlight conditions has not been studied, it is expected to be similar to that of 2,4-D, since the compounds are structurally similar. While Aly and Faust (1964) concluded that sunlight would not degrade 2,4-D, other authors (Crosby and Tutass, 1966; Boval and Smith, 1973; Binkley and Oakes, 1974) found evidence contrary to this conclusion. With both artificial and natural sunlight, Crosby and Tutass (1966) found that 2,4-D in water decomposed with the formation of 2,4-dichlorophenol, 4-chlorocatechol, 2-hydroxy-4-chlorophenoxyacetic acid and polymeric humic acids. In the case of dichloroprop, 2-hydroxy-4-chlorophenoxy propionic acid is expected to be produced in place of 2-hydroxy-4-chlorophenoxy acetic acid, as a result of photolysis. Zepp et al. (1975) estimated the photolytic half-life of 2,4-D butoxyethyl ester under sunlight irradiation conditions to be 12-14 days. The half-life for the methyl ester under the same conditions was estimated to be about twice as long.

Biodegradation of dichloroprop in aquatic systems may be a major process in the removal of this chemical. In the case of 2,4-D, several authors have shown that both isolated microorganisms and mixed cultures, such as those existing in natural aquatic systems, may biodegrade this compound (U.S. EPA,

1981). The biodegradation rate has been shown to depend on water temperature, pH, organic matter content, acclimation of microorganisms in water, availability of O_2 , the presence of suitable microorganisms and the population of microorganisms. Depending on the conditions of the aquatic media, the biodegradation half-life of 2,4-D may vary from 5-18 days (U.S. EPA, 1981). Unfortunately, similar biodegradation data are not available for dichloroprop. Based on the structural similarity of this compound to 2,4-D and the results of biodegradation study in soils (see Section 2.3.), the biodegradation of dichloroprop in natural aquatic media may be a significant degradation process.

The behavior of dichloroprop in water treatment with ozone was studied by Struif et al. (1978). Under normal conditions of water treatment with O_3 , dichloroprop was found to be partially oxidized to CO_2 , Cl^- , $(CO_2H)_2$, $CH_2OHCOOH$ and aliphatic aldehydes. The oxidizing effect of O_3 was found to increase with higher pH in the range of pH 6 to pH 9.

The losses of dichloroprop from aquatic media through evaporation and sedimentation of sorbed particulates have not been comprehensively studied. However, some estimate of the losses of dichloroprop through these physical processes can be made by analogy to these loss processes in the case of 2,4-D. The free acid form of 2,4-D, its sodium and potassium salts, and its triethanolamine salts have insignificant volatility (Bovey, 1980). The esters of 2,4-D have variable volatility. The vaporization half-lives for methyl, 1-butyl, 1-octyl and 2-butoxyethyl esters of 2,4-D have been estimated to be 21.7, 1.1, 11.5 and 895 days, respectively (Zepp et al., 1975).

From the studies of Aly and Faust (1964) with 2,4-D esters and the value of K_{oc} for dichloroprop (the K_{oc} for esters may be higher than the free acid because the esters have lower aqueous solubilities), it can be

predicted that dichloroprop may not be strongly sorbed to particulate matter in water. Of course, the sorption characteristics are likely to depend on the organic matter content of the water particulates, with the sorption increasing as the organic matter content increases. The investigation of Smith and Isom (1967) shows that some sedimentation of particle-bound dichloroprop from aquatic media is a likely process.

2.2. AIR

There is a lack of data in the literature concerning the atmospheric fate and transport of dichloroprop. Drawing an analogy from 2,4-D butyl ester (Que Hee and Suitherland, 1979), it can be predicted that the photolytic half-life of dichloroprop esters in the atmosphere will be ~13 days. The hydrolysis of dichloroprop esters also appears to be a likely process. The work of Grover et al. (1976) with 2,4-D esters also indicates that washouts of dichloroprop from the atmosphere by rain will occur; however, in the absence of more data, it is impossible to estimate the overall half-life of dichloroprop in the atmosphere.

2.3. SOIL

The two likely chemical processes that may significantly dictate the fate of dichloroprop and of its salts and esters in soils are hydrolysis and photodecomposition. Unfortunately, specific studies determining the fate of this chemical under both photolytic and hydrolytic conditions in soil have not been made. Based on the studies done on an analogous compound, 2,4-D (U.S. EPA, 1981), esters of dichloroprop can be predicted to undergo relatively rapid hydrolysis in moist soils, particularly with a rise in soil temperature. The hydrolysis of esters may be complete within 1-3 days. Although hydrolysis may be able to convert the esters into free acid, this process is not capable of directly degrading dichloroprop in soils.

The photochemical fate of dichloroprop in soil can be predicted from its behavior in aqueous media (see Section 2.1.) and from analogy to the fate of 2,4-D in soil (U.S. EPA, 1981). Photochemical degradation of dichloroprop in soils will probably take place only at the soil surface because of light attenuation at subsurface soil. Although no quantitative value is available, the half-life of dichloroprop on the soil surface may be predicted to be >15 days, based on analogy to the half-life of 2,4-D in aquatic media.

The most important process that may account for the significant loss of dichloroprop in soil is biodegradation. Several authors have studied the biodegradation of dichloroprop in soils. Burger et al. (1962) showed that a species of bacterium, Flavobacterium sp., can biodegrade dichloroprop. The biodegradability of dichloroprop with mixed bacterial cultures isolated from soil has been shown by Kilpi (1980). Smith (1978) estimated the biodegradation half-life of dichloroprop to be 8-12 days, depending on the soil characteristics. Hance (1979) reported that dichloroprop essentially disappeared from the soils in 14 days with two soils incubated at 22°C and 80% water holding capacity. This author did not observe an effect of pH in the biodegradation of dichloroprop in soil. Van Schreven et al. (1970) showed that the biodegradation is dependent on the initial concentration of dichloroprop in soils. When treated with a 100-fold greater than normal rate of dichloroprop, ~38% of the herbicide degraded in 9 months.

The loss of dichloroprop through the likely physical processes (leaching, runoff and volatilization) has not been studied comprehensively. From the simulated ryegrass, Lolium perenne, ecosystem study of Gile (1983), where 2,4-D butyl ester was used, it can be predicted that volatilization of dichloroprop esters is likely from soils. The study of Nicholaichuk and Grover (1983) determined that ~4% of applied 2,4-D can be lost from soil

during spring runoff from agricultural watersheds. Since dichloroprop has lower solubility and a higher K_{oc} value than 2,4-D (Kenaga, 1980), the runoff from dichloroprop is expected to be lower than in the case of 2,4-D. The ability of dichloroprop to move vertically through soil, causing leaching, may be dependent on the soil characteristics and the chemical form of dichloroprop. Consistent with the leachability study done with 2,4-D (U.S. EPA, 1981), it can be predicted that dichloroprop is not likely to leach significantly into groundwater.

Finally, the overall half-life of dichloroprop in soil has been estimated to be 10 days by Altom and Stritzke (1973) and ~8-10 days by Kirkland and Fryer (1972) with different kinds of treated and untreated soils. Therefore, this compound is not likely to persist in soil for a long period of time.

3. EXPOSURE

3.1. WATER

The U.S. EPA's STORET data base lists 4101 entries for the analysis of dichloroprop in whole water samples (water and sediment) and 13 entries for dissolved dichloroprop in water samples. Most of the water samples were collected from ambient streams. The mean concentration of dichloroprop in whole water was 0.05 $\mu\text{g}/\text{l}$, and the concentration ranged from none detected to 190 $\mu\text{g}/\text{l}$. The mean concentration for dissolved dichloroprop was 0.001 $\mu\text{g}/\text{l}$ with a range of none detected to 0.01 $\mu\text{g}/\text{l}$. No other monitoring data from dichloroprop in aquatic media are available.

3.2. AIR

Pertinent monitoring data regarding dichloroprop could not be located in the available literature.

3.3. FOOD

Data pertaining to the level of dichloroprop in a single food item or a composite food could not be located in the available literature.

4. PHARMACOKINETICS

4.1. ABSORPTION

That dichloroprop is absorbed from the GI tract of humans can be inferred from the case study reported by Prescott et al. (1979) of a 58-year-old woman who ingested a weedkiller (amount not reported) containing 21.4% dichloroprop (presumably as the acid form) and 16.1% 2,4-dichlorophenoxyacetic acid (2,4-D). Upon admission to the hospital (time after ingestion not specified), the plasma concentration of dichloroprop was 400 µg/mL. The plasma half-life of dichloroprop, measured over 5 days, was 95 hours.

4.2. DISTRIBUTION

Prescott et al. (1979) reported that a 58-year-old woman, who had ingested an unspecified amount of a weedkiller containing 21.4% dichloroprop (presumably as the acid form) and 16.1% 2,4-D, had plasma concentrations of 400 µg dichloroprop/mL upon admission to the hospital (time after ingestion not specified). Details regarding further distribution of dichloroprop were not reported.

4.3. METABOLISM

A 58-year-old woman who had ingested an unspecified amount of a weedkiller containing 21.4% dichloroprop (presumably as the acid form) 16.1% 2,4-D, excreted 70% of the ingested dichloroprop unchanged in the urine during the first 5 days after ingestion (Prescott et al., 1979). No mention was made of the remaining 30% of the dose.

4.4. EXCRETION

That urinary excretion of dichloroprop occurs can be inferred from the case study of a 58-year-old woman who had ingested an unspecified amount of a weedkiller containing 21.4% dichloroprop (presumably as the acid form) and 16.1% 2,4-D, in that 70% of the ingested dichloroprop was excreted intact in the urine within 5 days (Prescott et al., 1979).

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of dichloroprop could not be located in the available literature. Dichloroprop has not been scheduled for testing in a carcinogenesis bioassay by the National Toxicology Program (NTP, 1983).

5.2. MUTAGENICITY

Dichloroprop was not mutagenic to eight histidine-deficient strains of Salmonella typhimurium in the absence of a mammalian metabolic activation system (Andersen et al., 1972), but induced respiration-defective mutant cells of Saccharomyces cerevisiae (Schubert, 1969). These mutagenicity assays are summarized in Table 5-1.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of dichloroprop could not be located in the available literature.

5.4. OTHER REPRODUCTIVE EFFECTS

Following administration of dichloroprop intraperitoneally as a single dose of 200 mg/kg bw to male mice, Seiler (1979) measured resulting inhibition of testicular DNA synthesis as a function of depressed thymidine uptake. Treatment resulted in 14% inhibition of testicular DNA synthesis, which was not significantly different from control DNA synthesis.

5.5. CHRONIC TOXICITY

Pertinent data regarding the chronic or subchronic toxicity of dichloroprop could not be located in the available literature.

TABLE 5-1
Mutagenicity Testing of Dichloroprop

Assay	Indicator Organism	Application	Concentration or Dose	Activation System	Response*	Reference
Reverse mutation	<u>Salmonella typhimurium</u> 8 histidine-deficient strains, NR	spot test	-1-5 μ A/plate	none	.	Andersen et al., 1972
Induction of respiration-defective mutant cells	<u>Saccharomyces cerevisiae</u>	cell culture	0.7 or 3.0 mg/ml	none	+	Schubert, 1969

*Responses: +, positive; -, negative
NR = Not reported

5.6. OTHER RELEVANT INFORMATION

The oral LD₅₀s for dichloroprop have been reported to be 800 mg/kg in rats and 400 mg/kg in mice, while the dermal LD₅₀ has been reported to be 1400 mg/kg for both species (NIOSH, 1983). Senczuk and Pogorzelska (1980) reported an oral LD₅₀ of 309.4 mg/kg for dichloroprop in mice.

Following induction with dichloroprop (dose not reported in available abstract), Schiller et al. (1982) observed a 3-fold higher level of ethoxy-resorufin O-deethylase in the liver of suckling rats (10-day-old) as compared with adult rats (60-day-old). A dose-related increase in cytochrome P-450 concentration was seen in rats (age not specified in available abstract) induced with dichloroprop (Schiller et al., 1982).

Dichloroprop inhibited the activity of glutamic-dehydrogenase obtained from ox liver in vitro (Polemio et al., 1961).

6. AQUATIC TOXICITY

Data on the toxicity of dichloroprop to aquatic organisms are limited. In bluegill sunfish, Lepomis macrochirus, the static 48-hour LC_{50} was 1.1 mg dichloroprop/l (FWPCA, 1968). Schneider (1979), however, reported that all bluegills survived 96 hours of exposure at 180 mg dichloroprop/l (purity 95.6%) under static conditions. Rainbow trout, Salmo gairdneri, exposed at 180 mg/l survived up to 48 hours but had 20% mortality by 96 hours.

Additional data on the acute or chronic toxic effects of dichloroprop on aquatic animals or plants were not located in the available literature. No data on the accumulation or persistence of dichloroprop residues in aquatic organisms were found.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Pertinent data regarding the existing guidelines and standards for dichloroprop could not be located in the available literature.

7.2 AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of dichloroprop could not be located in the available literature.

8. RISK ASSESSMENT

The lack of carcinogenicity and chronic toxicity bioassays in the available literature precludes the derivation of a quantitative risk criterion for dichloroprop. Dichloroprop was mutagenic to Saccharomyces cerevisiae (Schubert, 1969), but not to Salmonella typhimurium in the absence of a mammalian metabolic activation system (Andersen et al., 1972). Seiler (1979) did not observe a statistically significant increase in inhibition of testicular DNA synthesis in male mice following administration of dichloroprop as a single intraperitoneal dose of 200 mg/kg bw. Inhibition of glutamic-dehydrogenase activity by dichloroprop has been observed in vitro (Polemio et al., 1981).

Statements concerning available literature in this document refer to published, quotable sources and are in no way meant to imply that confidential data, which this document could not address, are not in existence. These data, which are considered Confidential Business Information (CBI), are available in summary form as a CBI Appendix that can be accessed by individuals with appropriate clearance. Conclusions from the CBI Appendix regarding risk assessment can, however, be presented in this chapter as follows.

The CBI Appendix to this document reviews other animal data from which an ADI of 0.25 mg/kg bw/day or 17.5 mg/day for a 70 kg man was calculated. The ADI was based on a NOAEL of 25 mg/kg bw/day for reduced body weight gain and food intake in rats in a chronic feeding study and an uncertainty factor of 100 (10 to account for interspecies extrapolation and 10 for protection of the most sensitive individuals within the human population).

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APPENDIX
Literature Searched

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE

These searches were conducted in November, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7th), and the following secondary sources were reviewed:

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